Health Psychology
2000, Vol. 19, No. 1, 91-97
Jan.

DOI: 10.1037//0278-6133.19.1.91

Physician-Diagnosed Medical Disorders in Relation to PTSD Symptoms in Older Male Military Veterans

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The association between physician-diagnosed medical disorders and combat-related posttraumatic stress disorder (PTSD) symptoms was examined in 605 male combat veterans of World War II and the Korean conflict. Physician exams were performed at periodic intervals beginning in the 1960s. PTSD symptoms were assessed in 1990. Cox regression was used to examine the onset of each of 12 disorder categories as a function of PTSD symptoms, controlling for age, smoking, alcohol use, and body weight at study entry. Even with control for these factors, PTSD symptoms were associated with increased onset of arterial, lower gastrointestinal, dermatologic, and musculoskeletal disorders. There was only weak evidence that PTSD mediated the effects of combat exposure on morbidity. Possible mediators of the relationship between combat exposure, PTSD, and physical morbidity are discussed.

Key words: posttraumatic stress disorder, military veterans, medical disorders, aged

Exposure to traumatic events is related to poor physical health status. For example, Felitti et al. (1998) recently reported that adverse childhood experiences were associated with increased risk of physician-diagnosed ischemic heart disease, cancer, stroke, chronic bronchitis or emphysema, diabetes, and fractures among enrollees in a large health maintenance organization. In an attempt to understand how trauma relates to poor health, Wolfe, Schnurr, Brown, and Furey (1994) presented data showing that posttraumatic stress disorder (PTSD) was a potential mediator of the relationship between trauma and poor health in female veterans of the Vietnam War. Several studies have used path analysis to formally demonstrate the mediational role of PTSD in the relationship between trauma and physical health (Friedman & Schnurr, 1995; Schnurr & Spiro, 1999; Taft, Stern, King, & King, 1999).

Information about PTSD and physical health is relatively sparse, compared with the literature linking traumatic exposure and poor health outcomes. The majority of evidence is based on self-reports of health status or conditions (Friedman & Schnurr, 1995). In one of the largest of these studies, PTSD in male Vietnam veterans was associated with increased likelihood of self-reported circulatory, digestive, musculoskeletal, nervous system, and respiratory disorders, even after statistical control for factors such as age, hypochondriasis, and pack years of smoking (Boscarino, 1997).

Far less is known about the relationship between PTSD and morbidity, although the limited information available links PTSD to poor outcomes. Shalev, Bleich, and Ursano (1990) compared male Israeli combat veterans with and without PTSD and found the PTSD group had poorer effort tolerance in a laboratory stress test, even when smoking status was controlled. Boscarino and Chang (1999) examined electrocardiogram results in male Vietnam veterans with and without PTSD. While controlling for factors such as alcohol consumption, current substance abuse, smoking, and current medication use, the authors found that PTSD was associated with nonspecific abnormalities, conduction defects, and infarctions. Beckham et al. (1998) compared male Vietnam veterans with and without PTSD and found that the PTSD group had relatively more physician-diagnosed disorders, even after adjustment for factors such

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This research was supported by a Department of Veterans Affairs Merit Review grant. The Normative Aging Study is supported by the Department of Veterans Affairs' Cooperative Studies Program and is a component of the Massachusetts Veterans' Epidemiology Research and Information Center. Portions of the data were presented at the Annual Meeting of the International Society for Traumatic Stress Studies, Montreal, Quebec, Canada, November 1997. We thank Brad Dain and Anjana Sengupta for helpful comments on the data analyses.

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as pack years of smoking and alcohol abuse. PTSD and non-PTSD groups did not differ in terms of the correspondence between self-reported and physician-diagnosed disorders; kappas for both tended to be low to moderate.

Further studies with morbidity as an outcome are needed in order to understand the relationship between PTSD and physical health. To address this need, we used data from the Department of Veterans Affairs (VA) Normative Aging Study (NAS; Spiro, Schnurr, & Aldwin, 1997). The NAS is a longitudinal project begun in 1961 to study aging in 2,280 initially healthy men, 95% of whom were veterans. The men have been assessed every 3–5 years by interview and physical exam and periodically by questionnaire. Fifty-four percent of the World War II veterans and 19% of the Korean conflict veterans served in combat (Spiro, Schnurr, & Aldwin, 1994). PTSD prevalence is roughly 1% among the combat veterans, which is low in comparison with younger cohorts (Kulka et al., 1990) but comparable to the 2% prevalence observed in another community-residing sample in which older men comprised virtually all of the combat group (Norris, 1992).

The NAS provides an opportunity to study the relationship between PTSD and physical health in a non-treatment-seeking sample. In a recent study, we used path analysis to model relationships among combat exposure, PTSD symptoms, and health behaviors in relation to self-reported physical health (Schnurr & Spiro, 1999). Overall, the NAS men reported themselves to be roughly 0.5 standard deviation healthier than age-similar men. Combat exposure was related to poorer health, but this relationship was mediated almost completely through PTSD symptoms. PTSD symptoms, in turn, were related to poor health, and little of this relationship was mediated by health behaviors.

The present study extended this work by assessing the relationship between PTSD and physical health as diagnosed by a physician as opposed to self-report. We used survival analysis to examine the onset of 12 types of medical disorders in relation to PTSD symptoms. An advantage of survival analysis is that it permits the study of how long it takes for an event to occur (rather than occurrence or nonoccurrence) even if some events are censored, that is, they do not occur. Although our primary objective was to assess the relationship between PTSD and physical health, and although survival analysis does not lend itself to formal mediational analysis as well as path analysis or structural equation modeling, we also examined health behaviors as mediators of the relationship between PTSD and medical disorders, and PTSD as a mediator of the relationship between combat and physical health.

Method

Participants

Participants were drawn from the Boston VA NAS. At study entry, NAS men were of slightly higher socioeconomic status than the U.S. population (Bossé, Ekerdt, & Silbert, 1984), and they ranged in age from 22 to 82 years. Enrollment occurred from 1961 through 1968 (*Mdn* = 1966).

As of February 1990, 16% of participants had died, and 6% had been lost to attrition. The present study drew from respondents to a survey on military service that was sent to all 1,778 study participants who were alive and enrolled in the study at that time. Over 80% responded. The 1,210 respondents who had served in the military during either World War II or the Korean conflict were comparable to nonrespondents (Spiro et al., 1994). For the present study, we targeted the 629 respondents who reported combat exposure; 605 had complete data on the predictors described below.

Most of the men were White (98%; $n = 584$). At study entry, average age was 43.9 years ($SD = 5.7$). Most (89%; $n = 533$) had graduated high school, 57% ($n = 341$) were employed in blue-collar or service occupations, and the rest held white-collar jobs. The majority (85%; $n = 513$) served in the military during World War II, versus the Korean conflict. (A few participants were missing data on some variables.)

Measures and Procedure

War-zone exposure. At study entry, 84% ($n = 524$) of the sample reported combat exposure on a single dichotomous questionnaire item. The 1990 survey included Keane's Combat Exposure Scale (CES), which has very good reliability and validity (Keane et al., 1989); 79% ($n = 490$) reported combat exposure on this scale ($M = 14.4$, $SD = 10.2$, range = 1–41). The 1990 survey also included two items adapted from Elder and Clipp (1988) that pilot testing had indicated were useful for identifying NAS men who had traumatic experience related to service in a war zone: "Over how long a period did you serve under combat conditions or subject to enemy action?" and "Even if you were not directly in combat (e.g., a physician), how many times were you exposed to the outcomes of combat (wounded or dead people)?" Twenty-three participants (4%) who had not reported combat exposure on the other two measures indicated exposure on one or both of these items. An affirmative response to the single dichotomous measure or a nonzero score on either the CES or the two additional measures was used to indicate a history of war-zone exposure.

PTSD. PTSD symptom severity was assessed through the 1990 military survey with the Mississippi Scale for Combat-Related PTSD (Keane, Caddell, & Taylor, 1988). In this instrument, each of 35 symptoms is rated on a 5-point scale, yielding a possible range of 35–175; higher scores reflect higher PTSD symptom levels. Although the scale was developed on samples of Vietnam veterans, it performs well as a measure of PTSD in older veterans (Engdahl, Eberly, & Blake, 1996; Hyer, Summers, Boyd, Litaker, & Boudewyns, 1996). A cutpoint of 89 has been recommended for determining a probable diagnosis of current PTSD (Kulka et al., 1990). There is no suggested cutpoint for using Mississippi Scale scores to diagnose lifetime PTSD. However, the actual time frame for most items is either unspecified or lifetime ("since the military"), and Mississippi Scale scores are elevated among military veterans with past PTSD only (Keane et al., 1998) or lifetime (Schnurr, Friedman, & Rosenberg, 1993) PTSD.

To explore the Mississippi Scale as a measure of lifetime PTSD, we used data from 258 male Vietnam-theater veterans who took part in the clinical interview component of the National Vietnam Veterans Readjustment Study, a large epidemiological study of a nationally representative sample of Vietnam-theater and Vietnam-era veterans (Kulka et al., 1990). A Mississippi Scale score ≥ 89 yielded .66 sensitivity and .82 specificity for a lifetime diagnosis of PTSD based on the Structured Clinical Interview for *DSM–III–R* (Spitzer, Williams, Gibbon, & First, 1987). There were substantial mean differences between lifetime PTSD ($M = 97.8$) and no lifetime PTSD ($M = 70.0$) groups as well, $t(256) = 11.01$ $p < .001$. Thus, we feel confident that our measure of PTSD provides a reasonable assessment of lifetime PTSD status.

Health risk behaviors. We controlled for smoking, regular alcohol consumption, and body mass index (BMI), all assessed at study entry. Data were not available for each variable at the time of onset of each disorder. Smoking was assessed by the Cornell Medical Index (CMI; Brodman, Erdman, & Wolff, 1956), with a single dichotomous item regarding whether a respondent smoked one or more packs of cigarettes per day; 120 men endorsed this item. According to data available for 93 of the smokers at their most recent NAS exam, the average number of years smoked (any amount of smoking) was 40.4 (SD = 9.8); 30% of the 114 for whom smoking status was assessed at the most recent exam were still smoking. Regular alcohol consumption was assessed by a CMI item that asked whether a respondent regularly drank two or more alcoholic beverages per day; 69 men endorsed this item. We compared this item to a measure of problem drinking that was available for some participants (n = 458) from a questionnaire administered in 1992: two or more problems on the CAGE (Buchsbaum, Buchanan, Welsh, Centor, & Schnoll, 1992). Men who met our definition of regular drinking according to the single CMI item were 4.88 times more likely than men who did not meet our definition to be problem drinkers according to the CAGE (95% confidence interval [CI] = 2.61–9.12, $p < .001$). BMI—weight in kilogram/height in squared meters—was treated as a continuous variable.

Medical diagnoses. Medical conditions were diagnosed by NAS physicians according to the eighth revision of the International Classification of Disease system (ICDA–8; U.S. Department of Health, Education, & Welfare, 1977) during routine health exams performed for all participants. Initially, men were scheduled for an exam every 5 years until age 52 and every 3 years thereafter. Since 1985, exams have been scheduled every 3 years regardless of age. Diagnoses were based on clinical exam findings and laboratory tests. The average number of exams per participant was 8.42 (range = 2–11). Across categories, the mean number of years between onset and the 1990 PTSD assessment was 7.11 (range = 2.84–11.90).

We initially selected 15 ICDA–8 chronic disorder categories to examine, on the basis of the literature linking traumatic exposure to poor health outcomes (Friedman & Schnurr, 1995). Three categories we had hoped to include (central nervous system, cerebrovascular, and liver) were deleted because they were not prevalent enough to permit an adequate events-to-variables ratio in multivariate analysis. The remaining 12 categories are presented in Table 1, along with their corresponding ICDA–8 numeric codes and examples.

Table 1
ICDA–8 Diagnostic Categories Used as Outcomes

Category	ICDA–8 code	Example
Malignant cancer	140–209	Prostate cancer, skin cancer
Endocrine	240–258	Diabetes, myxedema
Cardiovascular		
Hypertensive	400–404	Malignant hypertension
Ischemic	410–414	Myocardial infarction, angina pectoris
Other	420–429	Endocarditis, symptomatic heart disease
Arterial	440–448	Peripheral vascular disease
Pulmonary	490–493	Emphysema, asthma
Gastrointestinal		
Upper	530–537	Peptic ulcer, duodenal ulcer
Lower	560–569	Diverticulitis, colitis, irritable bowel
Genitourinary	590–607	Benign prostatic hyperplasia
Dermatologic	690–698	Eczema, dermatitis, psoriasis
Musculoskeletal	710–718	Rheumatoid arthritis, osteoarthritis

Note. ICDA–8 = *Eighth Revision of the International Classification of Diseases, Adapted* (U.S. Department of Health, Education, and Welfare, 1977).

Data Analysis

Cox regression was used to model time to the first occurrence of disease following study entry for each of the 12 categories. Very few disorders were present at study entry because NAS men were selected for having good physical health; thus, <1% to 6% of the men were excluded from the analysis for each outcome (as indicated by the Ns in Table 2). Coefficients for PTSD symptoms and combat exposure are presented per 10-point increase and are adjusted for age at study entry, smoking, alcohol consumption, and BMI.

The hazard function (at a given time) in Cox regression is the rate at which an event occurs, given that an individual has survived until that time. The analysis is based on an assumption of *proportional hazards*—that the ratio of hazards is constant across time for levels of predictors. This assumption was tested for PTSD symptoms, a continuous predictor, by creating low-, moderate-, and high-symptom severity categories (<60, 60–79, and ≥80, respectively) and comparing log-minus-log survival plots for all outcomes for which PTSD was a significant predictor. Visual inspection indicated that PTSD met the assumption of proportional hazards.

Results

At study entry, 20% (n = 120) of the participants were regular smokers. Eleven percent (n = 69) reported regularly drinking at least two drinks per day. Mean BMI was 25.9 (SD = 2.7); 21% (n = 127) of the sample met the conventional definition of obesity (BMI ≥ 27.8). At the time of PTSD assessment, their average age was 67.7 (SD = 5.7). The mean PTSD score on the Mississippi Scale was low (59.6, SD = 11.0, range = 36–140). Six men (1%) had a diagnosis of PTSD according to the diagnostic cutpoint of 89; an additional 19 (3%) had scores between 80 and 88. The correlation of PTSD symptoms with combat exposure severity was .19 ($p < .001$).

PTSD symptoms were modestly correlated with the health risk variables. The point-biserial $r(605)$ for smoking and for alcohol consumption was .13 and .10 ($ps < .005$ and .05, respectively). The Pearson $r(605)$ between PTSD symptoms and BMI was .09 ($p < .05$).

Table 2 lists for each of the 12 disorder categories the percentage of participants who were diagnosed with an initial onset during study participation. The least frequent disorders, occurring in 12% of the participants, were lower gastrointestinal; the most frequent were genitourinary and arthritic, each occurring in roughly three quarters of the participants. Table 2 also contains the hazard ratios and 95% CIs for PTSD symptoms as predictors of each disorder category: first with adjustment only for age at study entry, and next with additional adjustment for smoking, alcohol consumption, and BMI. PTSD symptoms were associated with increased risk of four categories in both sets of

Table 2
Occurrence of ICDA–8 Diagnostic Categories as a Function of PTSD Symptoms in Male Veterans

Category	*N*	No. of events (%)	Hazard[a]	95% CI	Hazard[b]	95% CI
Malignant cancer	601	124 (20.6)	1.07	0.92–1.23	1.05	0.90–1.21
Endocrine	596	92 (15.4)	1.08	0.91–1.30	1.06	0.88–1.27
Cardiovascular						
Hypertensive	596	339 (56.9)	1.03	0.94–1.14	1.00	0.90–1.10
Ischemic	602	162 (26.9)	1.03	0.89–1.18	0.96	0.83–1.11
Other	592	303 (51.2)	0.92	0.83–1.02	0.92	0.83–1.03
Arterial	601	83 (13.8)	1.35***	1.14–1.61	1.27**	1.17–1.52
Pulmonary	596	112 (18.8)	1.05	0.89–1.22	0.95	0.80–1.13
Upper gastrointestinal	582	112 (19.2)	1.13	0.98–1.31	1.12	0.96–1.30
Lower gastrointestinal	590	70 (11.9)	1.23*	1.05–1.45	1.23*	1.04–1.45
Genitourinary	557	430 (77.2)	1.02	0.93–1.11	1.02	0.94–1.12
Dermatologic	581	172 (29.6)	1.16*	1.02–1.30	1.18*	1.05–1.34
Musculoskeletal	592	412 (69.6)	1.11**	1.03–1.20	1.09*	1.00–1.18

Note. Hazard rates are expressed per 10-point increase in PTSD scores. CI = confidence interval. ICDA–8 = *Eighth Revision of the International Classification of Diseases, Adapted* (U.S. Department of Health, Education, and Welfare, 1977). PTSD = posttraumatic stress disorder.
[a]Adjusted for age.
[b]Adjusted for age, smoking, alcohol consumption, and body mass index at study entry.
*$p < .05$. **$p < .01$. ***$p < .001$.

analyses. In the final models, for every 10-point increment in PTSD symptoms, risk increased 27% for arterial disorders, 23% for lower gastrointestinal disorders, 18% for dermatologic disorders, and 9% for musculoskeletal disorders. The effects of adjusting for health risk factors were small, but these factors independently increased risk in expected ways; for example, smoking increased the risk of pulmonary disorder by a factor of 3.5.

Figure 1 illustrates the association of PTSD symptoms with the onset of arterial disorders by displaying the cumulative survival function for three PTSD severity categories. As can be seen from the figure, by 30 years of participation in the study, 55% of the high-symptoms group had developed an arterial disorder, as compared with fewer than 20% of the low- and moderate-symptom severity groups.

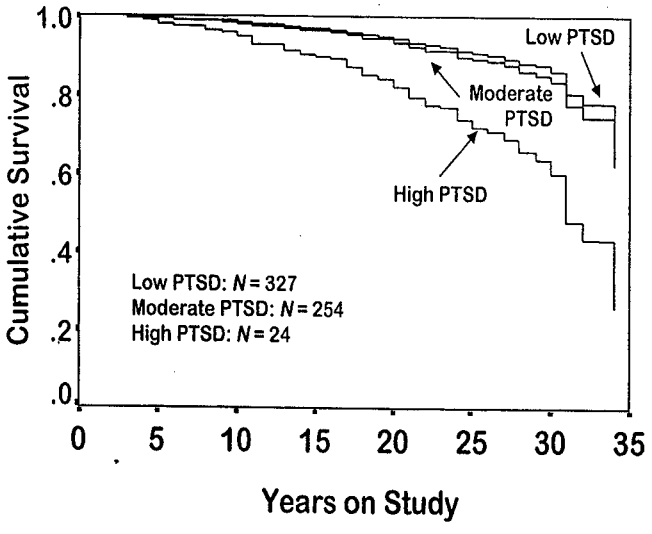

Figure 1. Occurrence of arterial disorders as a function of posttraumatic stress disorder (PTSD) symptom severity in male veterans. See text for definitions of low, moderate, and high PTSD.

To use the maximum amount of available data, we did not arbitrarily standardize the length of the observation period, which averaged 26.3 years but varied from 2.4 to 34.0 years. Although PTSD symptoms were uncorrelated with the length of the observation period, $r(605) = -.04, p = .30$, we examined the potential effects of variability on our findings by conducting the Cox regressions in a subsample of participants who had at least 20 years of observation (88%; $n = 535$). Not only did PTSD symptoms remain a significant predictor of each of the four disorder categories identified for the full sample, but the coefficients were remarkably similar to those observed for the full sample. The hazard rates from the fully adjusted analyses for arterial, lower gastrointestinal, dermatologic, and musculoskeletal disorders, respectively, were 1.35 ($p < .001$), 1.27 ($p < .005$), 1.18 ($p < .05$), and 1.08 ($p < .05$).

The role of PTSD symptoms in mediating the relationship between combat exposure and health outcomes was assessed by a hierarchical survival analysis for each disorder category. CES scores, age, and the other covariates were entered in an initial step, and PTSD symptoms were added in a second step. Amount of combat predicted increased onset of arterial, pulmonary, and upper gastrointestinal disorders; per 10-point increase in the CES, the hazard rate increased 1.23, 1.20, and 1.18, respectively (all $ps < .05$). Combat exposure also predicted decreased onset of other heart disorders (hazard = 0.87, $p < .05$). When PTSD was added to the models, only the effect for pulmonary disorders remained significant (hazard = 1.22, $p < .05$). However, the change in hazard rates for other disorders was small. The largest change was for arterial disorders, for which the hazard decreased to 1.18 ($p = .10$).

Discussion

We found that combat-related PTSD symptoms and combat exposure in older military veterans were associated with several types of medical disorders. In general, our results for PTSD parallel outcomes based on self-reported physical health in Vietnam veterans (Boscarino, 1997). Like Boscarino, we failed to find associations between PTSD and similarly defined cancer, genitourinary, and endocrine disorders. He also found associations between PTSD and respiratory and nervous system disorders, the latter of which we did not examine because of low prevalence. However, given the differences between samples in age, recruitment, war-zone exposure, and physician diagnosis (vs. self-report), the similarity of outcomes across the studies is striking.

Results for combat exposure are consistent with recent findings linking childhood trauma to physician-diagnosed chronic lung disease in adults (Felitti et al., 1998), and combat exposure to self-reported gastrointestinal and respiratory disorders in Vietnam-era twins (Eisen et al., 1998). The relationship of combat exposure to decreased onset of a mixed group of cardiovascular disorders is puzzling. It may be spurious given that there is no precedent in the literature to explain this finding and especially because combat was related to greater onset of other disorders.

The disorders found to be associated with PTSD symptoms and combat exposure in this study do not suggest a common etiology. Behavioral factors may be important, even though studies that have controlled for their effects (Beckham et al., 1998; Boscarino, 1997; Boscarino & Chang, 1999; the present study) or have modeled the mediational role of health behaviors (Schnurr & Spiro, 1999) have found substantial effects of PTSD on physical health. Most likely, the causal mechanisms are multifactorial. Friedman and Schnurr (1995) proposed that poor health could result from correlates of PTSD, for example, hyperreactivity, depression, and alcohol use. These correlates also could interact; for instance, alcohol use could result from an attempt to reduce hyperreactivity. Researchers need to examine these and other mechanisms to explain the association between PTSD and physical health and to determine whether any mechanisms are unique to PTSD. One such possibility is a pattern of hypothalamic-pituitary-adrenocortical axis function (low cortisol and enhanced receptor sensitivity) that is essentially opposite to the pattern observed in depression (Schnurr & Jankowski, 1999).

Analyses provided only limited support for PTSD as a mediator of the relationship between combat exposure and medical disorder. Although adjustment for PTSD caused statistically significant effects to become nonsignificant, the absolute change was small. Also, except for arterial disorders, the data failed to meet established criteria for testing mediation through hierarchical analysis (Baron & Kenney, 1986), which would have required both combat and PTSD to be associated with a given disorder. A possible reason for our findings is that 21% of our sample had reported combat only on measures other than the scale used in these analyses, which may have attenuated our ability to detect relationships that actually exist. Another reason is the modest correlation between amount of combat exposure and PTSD. Furthermore, we were not able to examine the effects of noncombat trauma because we did not have a measure of PTSD due to nonmilitary events. However, the most likely reason for our findings is the small number of men with clinically elevated PTSD symptoms. If the effect of trauma (combat) is mediated through one's reaction to the trauma (PTSD), the strength of mediation should be related to the extent of PTSD in a sample. The effect of combat through PTSD may have been quite diluted among these men. The health of the sample could also explain the .19 correlation between combat and PTSD symptoms; in contrast, Keane et al. (1989) found a correlation of .43 for the same measures in a non-PTSD sample.

PTSD was associated with only one of the four cardiovascular conditions studied. This was unexpected given that hyperarousal and hyperreactivity are presumed to result in greater risk of cardiovascular disorder in PTSD (Rosen & Fields, 1988). Laboratory studies have consistently found increased reactivity in PTSD and usually find higher resting heart rate (e.g., Keane et al., 1998). Future studies should determine the type of disorders that are related to PTSD and examine PTSD in relation to disease incidence and to severity. Future studies should also examine the mechanisms through which trauma might lead to poor outcomes. Longitudinal research is expensive, but one economical approach is to follow up a well-characterized sample of veterans, such as the National Vietnam Veterans Readjustment Study (Kulka et al., 1990).

It is premature to draw firm conclusions from the present study about the relationship of combat and PTSD to cardiovascular and other disorders. The initial selection of NAS men for good physical and mental health at study entry and their current good health (Schnurr & Spiro, 1999) limited generalization of findings to relatively healthy older veterans. Including only participants who survived long enough to have participated in our 1990 military survey also limited the generalizability. These factors, along with the small number of participants who had clinically elevated PTSD scores, may have attenuated our ability to detect relationships between PTSD symptoms and health outcomes.

Another methodological issue is that PTSD was not measured at study outset but in 1990, after many of the outcomes it was used to predict had accurred. It is implausible that combat-related PTSD resulted from the health problems studied in this article, but we addressed this issue by (a) confining the assessment to combat-related PTSD because combat occurred before the observation period, (b) using a measure with items that span the entire posttraumatic period as a time frame, and (c) demonstrating the sensitivity of our measure to lifetime PTSD in a different sample. Still, we may have underestimated lifetime PTSD severity. It is unlikely that this inflated our likelihood of finding a relationship between PTSD and morbidity, but future research should attempt to assess the status of PTSD and other covariates at the onset of each medical outcome.

This study is one of very few to document an association between PTSD and physical morbidity. The rarity of such

studies is significant because symptom exaggeration could substantially explain the relationship between PTSD and self-reported health. Exaggeration is an unlikely explanation for the results in our non-treatment-seeking sample, especially because they self-report above-average health (Schnurr & Spiro, 1999). However, a focus on carefully diagnosed morbidity is important in studies of treatment-seeking samples, for which there may be secondary gain in reporting poor health.

The negative health outcomes associated with trauma and PTSD have important public health implications, given that over half of the adult population in the United States have experienced a traumatic event and 8% have had PTSD at some point (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Trauma and PTSD could underlie a substantial amount of morbidity and utilization, and thus, cost.

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